

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF ILLINOIS

PREFERRED CAPITAL FUNDING,
NEVADA, LLC,

Plaintiff,

v.

Case No. 1:19-cv-06245
District Court Judge Virginia M. Kendall

JEFF KAHN, PHILLIP TIMOTHY HOWARD and
HOWARD & ASSOCIATES, P.A.,

Defendants.

**DEFENDANTS' PHILLIP TIMOTHY HOWARD AND HOWARD &
ASSOCIATES, P.A., MOTION TO DISMISS; MOTION FOR A MORE
DEFINITE STATEMENT; AND MOTION TO STRIKE**

COME NOW Defendants Phillip Timothy Howard, and Howard & Associates, P.A., (hereinafter "Defendants") through undersigned, pursuant to Federal Rule of Civil Procedure 12(b), (e) and (f), submits the following Motion to Dismiss, Motion for a More Definite Statement; and Motion to Strike, and as grounds would assert:

MOTION TO DISMISS

1. Lack of Subject-Matter Jurisdiction.

Defendants are not parties to any contract or agreement with Plaintiff, nor have Defendants provided independent medical opinion or independent medical information to Plaintiff upon which it would rely upon for determining to loan funds.

Plaintiffs are not lending institutions, Plaintiffs are not physicians, nor NFL Concussion Settlement Claims qualified MAF or BAP qualified physicians, nor are they risk and lending advisors to lending institutions.

2. Lack of Personal Jurisdiction.

Defendant Howard & Associates, P.A., does not exist as a law firm, never has existed, and is not a proper party.

3. Improper Venue.

Defendants are residents of Florida and New Jersey, and Plaintiff is a Nevada Company. Proper venue is not in Illinois.

4. Failure to State a Claim Upon Which Relief Can Be Granted.

Defendants are not parties to the contracts with their borrowers. Defendants are not the physicians, nor neurologists that evaluate brain injuries. Defendants did not make the risk and investment analysis upon which lender determined to enter into contracts with borrowers. Defendants are not the NFL Concussion Settlement Claims administrator for the NFL Concussion Settlement Claims, which claims administrator can change standards and procedures as accepted and guided by the magistrate and Federal District Court Judge Brody.

Plaintiff's borrowers will be in the process of advancing their claims for up to 65 years. Consistent with the 99% verification of CTE in every former NFL brain tested to date. The JAMA article by the Boston University Researchers found that

“CTE was neuropathologically diagnosed in . . . 110 out of 111 former NFL National Football League players (99%).” Thus, virtually all claimants will be paid over the life of the program. (July 25, 2017 JAMA Article attached as Exhibit A). In fact, in just the past few months, additional claims listed by Plaintiff has received NFL Concussion Settlement Claims Administrator award letters in the multiple \$ millions of dollars, and to date total approximately \$10 million in awards. In addition, there are 10 claims that are either awaiting testing results or are in the claims submission process with no eligibility determination yet made. Note, six of the Preferred Capital borrowers listed are no longer clients of Defendants. These details can be provided in an *in camera* or confidential and client protected review. Regardless of receiving an award letter, most large dollar claims are then appealed by the NFL, addressing items such as posting on social media, volunteering for sports teams, vacations with family, and more, and these outcomes are outside of any medical diagnosis provided by Dr. Koberda.

Dr. J. Lucas Koberda is indeed qualified by the NFL Settlement Claims Administrator for both MAF and BAP evaluations and provided the medical evaluations as of the date that they were given. Cumulative Exhibit B. The NFL Claims Administrator engaged in auditing approximately 56% of all claims filed by all parties and this delayed claims processing for approximately one year.

Howard has no management nor control over any Cambridge entity. Howard not only did not receive funds, he has well \$1.3 million invested into Cambridge.

Plaintiff's allegations that bringing in co-counsel to assist in advancing claims has harmed recoveries for clients is irrelevant and incongruous. Moreover, it is not the case. Bringing in expertise in managing claims is exactly what counsel for clients is required to do. Participating with experienced counsel that has a track record of success with the NFL Concussion Settlement Claims administrator is the type of action that protects the interests of NFL Concussion Settlement Claim clients and consequently the interests of the Plaintiff and logically cannot be the basis of a Complaint.

5. Failure to Join and Indispensable Party.

NFL Concussion Claims Administrator MAF and BAP qualified physician Dr. Lucas Koberda provided the medical evaluations of the borrowers, as applicable at that time. If Defendants are challenging the legitimacy of his medical reports, he is an indispensable and necessary party to this action, as he would necessarily be involved in any misrepresentation. If there is no misrepresentation in his reports, then Plaintiff does not have a valid action. NFL Concussion Claims Administrator determines and changes the concussion settlement protocols, not Defendants. They are an indispensable party to claims that the settlement protocols are invalid or fraudulently changed.

MOTION FOR A MORE DEFINITE STATEMENT

Pursuant to Rule 12(e), Federal Rules of Civil Procedure, a “party may move for a more definite statement of a pleading to which a responsive pleading is allowed but which is so vague or ambiguous that the party cannot reasonably prepare a response.” In this case, Plaintiff admits that to participate in the NFL Concussion Settlement Claims process that retired NFL players “must obtain a diagnosis by a previously approved NFL provider defined as a Baseline Assessment Provider (“BAP”) provider or a Monetary Award Fund (“MAF”) physician (board certified neurologist, board-certified neurosurgeon, or board-certified neuro-specialist who is a part of an approved list of providers).” Complaint, paragraph 17.

Plaintiff next claims Dr. Koberda is not “NFL Settlement approved.” Complaint, paragraph 52. This is demonstrably false. Dr. Koberda was and is NFL Concussion Settlement approved for both the MAF and BAP. (Cumulative Exhibit B)

Plaintiff claims that Dr. Koberda’s reports are fraudulent, but does not explain how Dr. Koberda, who is NFL Concussion Claim Administrator approved as both MAF and BAP NFL Concussion Claims diagnoses, and his reports and qualifications, are fraudulent. Without fraudulent medical reports, there is no viable action. Moreover, there is a logical impossibility for his medical reports to be false since they are subject to the evolving standards of the NFL Concussion Settlement

Claims process as determined by Judge Brody and the claims administrator. Thus, there is no substantive claim of his fraudulent activity, nor is Dr. Koberda a party to this action. There must be a more definite statement of Dr. Koberda's fraudulent activity in order for there to be a valid Complaint, or to respond to the Complaint.

Plaintiff does not list a date for the phone conversation, what was said, or what documents were provided. If the documents provided show that Dr. Koberda found that the retired NFL player had a 1.5 or a 2.0, then by definition, at that time, they would preliminarily meet the NFL Concussion Claims Settlement recovery standards. They would not necessarily receive an award, since the certifications of the underlying reports, years played, appeals, changes in protocols and more impact a final award payment.

The NFL Concussion Settlement Claims facility didn't open until March 23, 2017, and the BAP didn't start until June 6, 2017. Claims for the law firm clients were not filed until May of 2018 and afterwards, in order to ensure that a full understanding of the ever-evolving claims process and its changes were understood. Moreover, at that time Dr. Koberda's reports were updated consistent with the evolving protocols of the NFL Settlement Claims Administrator. The importance of updating reports is critical, in fact, by updating claims the thorough preparation and review has been increasing claim values from a 1.5 to a 2.0 or to Parkinson's from a 2.0 (Note injuries have increased in some clients, including those listed by Plaintiff,

from a 1.5 to a 2.0 and from a 2.0 to Parkinsons) or Dementia rating, and updating the years played.

Plaintiff does not explain how a delay by the NFL Concussion Settlement Claims administrator, even if for years, is the responsibility of Defendants? The reality is that regardless of the validity of an NFL Concussion Settlement Claim, Claims can languish in the NFL Concussions Settlement process for a year or more, and this has happened to hundreds of claims. Protocols for claims and medical diagnoses and reviews are constantly being changed and modified by the NFL Claims Administrator, and this requires constant vigilance. NFL Claims Administrator has audited of approximately 56% of all claims submitted, delaying processing of claims for over one year. Even claims not in audit and submitted can languish for a year or more. To date, only 2,902 claims out of 20,548 registered claimants have even submitted claims, and out of those 984 have received award notices, with our legal team having received a large percentage of those awards. (Exhibit C, NFL Concussion Settlement Report, October 14, 2019).

MOTION TO STRIKE

In violation of Rule 12 (f), Federal Rules of Civil Procedure, Plaintiff claims redundant, immaterial, impertinent and scandalous matters. Plaintiff claims fraud without any evidence or allegation that would amount to fraud. Plaintiff admits that Dr. Koberda provided the medical reports. Plaintiff admits that the physician has to

be MAF or BAP certified, which Dr. Koberda is both. Thus, claims of fraud are logically impossible and must be stricken.

Plaintiff claims that Howard solicited funds from players and received fees and didn't pay back loans. These allegations are false and Defendants will submit evidence demonstrating the same.

Plaintiff next claims that Defendants falsely presented that Dr. Koberda was NFL Settlement Approved. Complaint, paragraph 52. Dr. Koberda is NFL Settlement Approved. (Cumulative Exhibit B). Thus, the entire action, including the fraud claim, must be stricken.

Plaintiff claims that Dr. Koberda made "false diagnosis." Complaint, paragraph 53. This is false and Dr. Koberda's continued participation as a MAF and BAP qualified physician documents the same. (Cumulative Exhibit B).

Plaintiff finally claims that Dr. Koberda's reports were false and Defendants' knew this. This is logically impossible since Dr. Koberda's reports were true and still are. Moreover, Dr. Koberda is still a MAF and BAP qualified physician. *Id.*

MEMORANDUM OF LAW

Frivolous Action—Lack of Subject Matter Jurisdiction and Failure to State a Claim Upon Which Relief Can Be Granted.

Plaintiff's action claiming Dr. Koberda is not an NFL Concussion Settlement Claim MAF and BAP authorized neurologist is demonstrably false and frivolous. This is the core of Plaintiff's allegations—that Dr. Koberda's reports are false and

that he is not an NFL Concussion Settlement Claim MAF and BAP authorized neurologist. Plaintiff can prove no set of facts in support of its claim that would entitle it to relief. Although, on Fed. R. Civ. P. 12(b) motion to dismiss, court must look at all facts in light most favorable to plaintiff, if plaintiff can prove no set of facts which would entitle her to relief, her claims must be dismissed. 92 A.F.T.R.2d (RIA) 7186, 2003-2 U.S. Tax Cas. (CCH) P 50728.

The standard, that district court may not dismiss complaint for failure to state claim unless it appears beyond doubt that plaintiff can prove no set of facts in support of his claims which would entitle him to relief, often cited in Fed. R. Civ. P. 12(b)(6) motions, is equally applicable in Fed. R. Civ. P. 12(b)(1) motions challenging subject matter jurisdiction when such jurisdiction may be contingent upon factual matters in dispute. Mason v. Ariz., 260 F. Supp. 2d 807 (D. Ariz. 2003).

Thus, this action is frivolous and must be dismissed. *See* Rogers v. Stratton Industries, Inc., 798 F.2d 913, 41 Empl. Prac. Dec. (CCH) P36555, 41 Fair Empl. Prac. Cas. (BNA) 1160 (6th Cir. 1986) (In action alleging age discrimination in employment, plaintiff employee's affidavit, filed in response to employer's motion to dismiss for lack of subject matter jurisdiction, was insufficient to raise genuine issue as to material fact, and complaint was properly dismissed under Rule 12(b)(1) or 12(b)(6). Where motions to dismiss are based on both subject matter jurisdiction and personal jurisdiction, in accordance with Supreme Court precedent, subject

matter jurisdiction should be addressed before personal jurisdiction. Gadlin v. Sybron Int'l Corp., 222 F.3d 797, 2000 Colo. J. C.A.R. 4750 (10th Cir. 2000).

Whether it is termed motion to dismiss for lack of jurisdiction over subject matter or for failure to state claim upon which relief can be granted, result of such motion, if granted, is same. Hospital Bldg. Co. v. Trustees of Rex Hospital, 511 F.2d 678, 1974 Trade Cas. (CCH) P74903, 1975-1 Trade Cas. (CCH) P60166 (4th Cir. 1975), *rev'd*, 425 U.S. 738, 96 S. Ct. 1848, 48 L. Ed. 2d 338, 21 Fed. R. Serv. 2d (Callaghan) 845, 1976-1 Trade Cas. (CCH) P60885 (1976).

Incontrovertible Evidence Presented in Support of Frivolous Action.

Moreover, the evidence presented in the Motion to Dismiss, such as Dr. Koberda's verification that he is an authorized MAF Physician on the NFL Concussion Settlement Claims website, that 99% of NFL Players will have CTE, and the affidavit documenting the invalidity of statements by Plaintiffs, is valid for the Court to consider in dismissing the action. Facts alleged in motion need not be sworn to or contained in affidavit, and use of affidavits under such circumstances is discretionary. Victory v. Manning, 128 F.2d 415, 29 A.F.T.R. (P-H) 518 (3d Cir. 1942). *See also* English v. Cowell, 10 F.3d 434, 27 Fed. R. Serv. 3d (Callaghan) 513, 144 L.R.R.M. (BNA) 2754, 126 Lab. Cas. (CCH) P10928 (7th Cir. 1993).

Though “matters outside pleading” may not be considered in deciding Fed. R. Civ. P. 12 motion to dismiss, documents necessarily embraced by Complaint are not matters outside pleading. Enervations, Inc. v 3M 380 F.3d 1066 (CA8 Minn 2004).

Transcript and news segment were not “matters outside pleadings” because they were both documents to which complaint had referred, documents were concededly authentic, and they were central to plaintiffs’ claim. Santana v. Cook County Bd. of Review, 679 F.3d 614 (7th Cir. 2012).

Affidavits may be considered on motion to dismiss raising objections bearing on question of jurisdiction, venue, or process. Kentucky-Tennessee Light & Power Co. v. Nashville Coal Co., 37 F. Supp. 728 (D. Ky. 1941).

Affidavits, depositions, and other documentary proof may be utilized when movant seeks dismissal of case upon any of first 5 defenses set forth in Rule 12(b), because very nature of those defenses is such as to admit of proof by *ex parte* statements in most instances, but where defense of failure to state claim upon which relief can be granted is relied upon, court should determine motion upon allegations of complaint and undisputed facts as they appear from pleadings, orders, and records of case. Yudin v. Carroll, 57 F. Supp. 793 (D. Ark. 1944).

Lack of Jurisdiction and Improper Party.

Since Plaintiff has attempted to sue and serve Howard & Associates, P.A., which is a company that does not exist as a law firm in Florida, it is elementary that

if court has no jurisdiction over defendant, and defendant has unqualified right to have order entered granting its motion to dismiss. Read v. Ulmer, 308 F.2d 915, 8 Av. Cas. (CCH) P17200, 6 Fed. R. Serv. 2d (Callaghan) 766 (5th Cir. 1962)

WHEREFORE, for the foregoing reasons, Defendants request that this Court dismiss this Complaint as frivolous and impossible to state a claim, or in the alternative require proper parties, proper subject matter, proper jurisdiction, proper allegations, provide a more definite statement, and strike scandalous and logically impossible claims.

Respectfully submitted on this 18th Day of October, 2019.




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CERTIFICATE OF SERVICE

I hereby certify that on this 18th day of October 2019, a copy of the foregoing was served by email to the following counsel of record:

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Tim Howard, Esq.,

EXHIBIT A

Original Investigation

July 25, 2017

Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football

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Author Affiliations Article Information
JAMA. 2017;318(4):360-370. doi:10.1001/jama.2017.8334

Key Points

Question What are the neuropathological and clinical features of a case series of deceased players of American football neuropathologically diagnosed as having chronic traumatic encephalopathy (CTE)?

Findings In a convenience sample of 202 deceased players of American football from a brain donation program, CTE was neuropathologically diagnosed in 177 players across all levels of play (87%), including 110 of 111 former National Football League players (99%).

Meaning In a convenience sample of deceased players of American football, a high proportion showed pathological evidence of CTE, suggesting that CTE may be related to prior participation in football.

Abstract

Importance Players of American football may be at increased risk of long-term neurological conditions, particularly chronic traumatic encephalopathy (CTE).

Objective To determine the neuropathological and clinical features of deceased football players with CTE.

Design, Setting, and Participants Case series of 202 football players whose brains were donated for research. Neuropathological evaluations and retrospective telephone

clinical assessments (including head trauma history) with informants were performed blinded. Online questionnaires ascertained athletic and military history.

Exposures Participation in American football at any level of play.

Main Outcomes and Measures Neuropathological diagnoses of neurodegenerative diseases, including CTE, based on defined diagnostic criteria; CTE neuropathological severity (stages I to IV or dichotomized into mild [stages I and II] and severe [stages III and IV]); informant-reported athletic history and, for players who died in 2014 or later, clinical presentation, including behavior, mood, and cognitive symptoms and dementia.

Results Among 202 deceased former football players (median age at death, 66 years [interquartile range, 47-76 years]), CTE was neuropathologically diagnosed in 177 players (87%; median age at death, 67 years [interquartile range, 52-77 years]; mean years of football participation, 15.1 [SD, 5.2]), including 0 of 2 pre-high school, 3 of 14 high school (21%), 48 of 53 college (91%), 9 of 14 semiprofessional (64%), 7 of 8 Canadian Football League (88%), and 110 of 111 National Football League (99%) players. Neuropathological severity of CTE was distributed across the highest level of play, with all 3 former high school players having mild pathology and the majority of former college (27 [56%]), semiprofessional (5 [56%]), and professional (101 [86%]) players having severe pathology. Among 27 participants with mild CTE pathology, 26 (96%) had behavioral or mood symptoms or both, 23 (85%) had cognitive symptoms, and 9 (33%) had signs of dementia. Among 84 participants with severe CTE pathology, 75 (89%) had behavioral or mood symptoms or both, 80 (95%) had cognitive symptoms, and 71 (85%) had signs of dementia.

Conclusions and Relevance In a convenience sample of deceased football players who donated their brains for research, a high proportion had neuropathological evidence of CTE, suggesting that CTE may be related to prior participation in football.

Introduction

Chronic traumatic encephalopathy (CTE) is a progressive neurodegeneration associated with repetitive head trauma.¹⁻⁸ In 2013, based on a report of the clinical and pathological features of 68 men with CTE (including 36 football players from the current study), criteria for neuropathological diagnosis of CTE and a staging scheme of pathological severity were proposed.⁶ Two clinical presentations of CTE were described; in one, the initial features developed at a younger age and involved behavioral disturbance, mood disturbance, or both; in the other, the initial presentation developed at an older age and involved cognitive impairment.⁹ In 2014, a methodologically rigorous approach to assessing clinicopathological correlation in CTE was developed using comprehensive structured and semistructured informant interviews and online surveys conducted by a team of behavioral neurologists and neuropsychologists.¹⁰ In 2015, the neuropathological criteria for diagnosis of CTE were refined by a panel of expert neuropathologists organized by the National Institute of

Neurological Disorders and Stroke and the National Institute of Biomedical Imaging and Bioengineering (NINDS-NIBIB).⁸

Using the NINDS-NIBIB criteria to diagnose CTE and the improved methods for clinicopathological correlation, the purpose of this study was to determine the neuropathological and clinical features of a case series of deceased football players neuropathologically diagnosed as having CTE whose brains were donated for research.

Methods

Study Recruitment

In 2008, as a collaboration among the VA Boston Healthcare System, Bedford VA, Boston University (BU) School of Medicine, and Sports Legacy Institute (now the Concussion Legacy Foundation [CLF]), a brain bank was created to better understand the long-term effects of repetitive head trauma experienced through contact sport participation and military-related exposure. The purpose of the brain bank was to comprehensively examine the neuropathology and clinical presentation of brain donors considered at risk of development of CTE. The institutional review board at Boston University Medical Campus approved all research activities. The next of kin or legally authorized representative of each brain donor provided written informed consent. No stipend for participation was provided. Inclusion criteria were based entirely on exposure to repetitive head trauma (eg, contact sports, military service, or domestic violence), regardless of whether symptoms manifested during life. Playing American football was sufficient for inclusion. Because of limited resources, more strict inclusion criteria were implemented in 2014 and required that football players who died after age 35 years have at least 2 years of college-level play. Donors were excluded if postmortem interval exceeded 72 hours or if fixed tissue fragments representing less than half the total brain volume were received (eFigure in the [Supplement](#)).

Clinical data were collected into a Federal Interagency Traumatic Brain Injury Research-compliant database. Since tracking began in 2014, for 98 (81%) brain donations to the VA-BU-CLF Brain Bank, the next of kin approached the brain bank near the time of death. The remaining brain donors were referred by medical examiners (11 [9%]), recruited by a CLF representative (7 [6%]), or participated in the Brain Donation Registry during life (5 [4%]) (eFigure in the [Supplement](#)).

Clinical Evaluation

Retrospective clinical evaluations were performed using online surveys and structured and semistructured postmortem telephone interviews between researchers and informants. Researchers conducting these evaluations were blinded to the neuropathological analysis, and informants were interviewed before receiving the results of the neuropathological examination. A behavioral neurologist, neuroscientist, or neuropsychologist (J.M., D.H.D., T.M.S., M.L.A., or R.A.S.) obtained a detailed history, including a timeline of cognitive, behavioral, mood, and motor symptomology.

Additionally, other neuropsychiatric symptoms, exposures and symptoms consistent with posttraumatic stress disorder, features of a substance use disorder, neurodegenerative diagnoses made in life (Alzheimer disease [AD], frontotemporal dementia, vascular dementia, dementia with Lewy bodies, Parkinson disease, CTE, or dementia of unknown etiology), headaches that impaired function, symptoms and diagnoses made in life of sleep disorders, and causes of death were assessed. Clinicians qualitatively summarized the participants' clinical presentation (eg, presence and course of symptoms, functional independence) into a narrative and presented the case to a multidisciplinary consensus team of clinicians, during which it was determined whether the participant met criteria for dementia. To resolve discrepancies in methods that evolved over time, only clinical variables ascertained after January 2014 using a standardized informant report were included because of the larger subset of participants recruited during this time frame (n=125).

Prior to January 2014, demographics, educational attainment, athletic history (type of sports played, level, position, age at first exposure, and duration), military history (branch, location of service, and duration of combat exposure), and traumatic brain injury (TBI) history (including number of concussions) were queried during the telephone interview. Beginning in January 2014, demographics, educational attainment, and athletic and military history were queried using an online questionnaire. Informant-reported race was collected as part of demographic information so that neuropathological differences across race could be assessed. To be considered a National Football League (NFL) athlete, a participant must have played in at least 1 regular-season NFL game. Professional position and years of play were verified using available online databases (<http://www.pro-football-reference.com>, <http://databasefootball.com>, <http://www.justsportsstats.com>). History of TBI was queried using informant versions of the Ohio State University TBI Identification Method Short Form¹¹ and 2 questionnaires adapted from published studies that address military-related head injuries and concussions.^{12,13} With the addition of these questionnaires, informants were read a formal definition of concussion prior to being asked about concussion history, which was not the case prior to January 2014.

Neuropathological Evaluation

Pathological processing and evaluation were conducted using previously published methods.^{14,15} Brain volume and macroscopic features were recorded during initial processing. Twenty-two sections of paraffin-embedded tissue were stained for Luxol fast blue, hematoxylin and eosin, Bielschowsky silver, phosphorylated tau (ptau) (AT8), α -synuclein, amyloid- β , and phosphorylated transactive response DNA binding protein 43 kDa (pTDP-43) using methods described previously.¹⁶ In some cases, large coronal slabs of the cerebral hemispheres were also cut at 50 μ m on a sledge microtome and stained as free-floating sections using AT8 or CP-13.^{16,17}

A neuropathological diagnosis was made using criteria for CTE recently defined by the 2015 NINDS-NIBIB Consensus Conference⁸ and well-established criteria for other neuropathological diseases, including AD,^{18,19} Lewy body disease,²⁰ frontotemporal lobar degeneration,²¹⁻²⁵ and motor neuron disease.^{26,27} Neuropathological criteria for CTE require at least 1 perivascular tau lesion consisting of tau aggregates in neurons, astrocytes, and cell processes around a small blood vessel; these pathognomonic CTE lesions are most often distributed at the depths of the sulci in the cerebral cortex and are distinct from the lesions of aging-related tau astroglialopathy.⁸ Supportive features for the diagnosis of CTE include tau pretangles and neurofibrillary tangles (NFTs) in superficial cortical layers (layers II/III) of the cerebral cortex; pretangles, NFTs or extracellular tangles in CA2 and CA4 of the hippocampus; subpial tau astrocytes at the glial limitans; and dot-like tau neurites.⁸

Chronic traumatic encephalopathy tau pathology was classified into 4 stages using previously proposed criteria.⁶ Briefly, stage I CTE is characterized by 1 or 2 isolated perivascular epicenters of tau NFTs and neurites (ie, CTE lesions) at the depths of the cerebral sulci in the frontal, temporal, or parietal cortices. In stage II, 3 or more CTE lesions are found in multiple cortical regions and superficial NFTs are found along the sulcal wall and at gyral crests. Multiple CTE lesions, superficial cortical NFTs, and diffuse neurofibrillary degeneration of the entorhinal and perirhinal cortices, amygdala, and hippocampus are found in stage III CTE. In stage IV CTE, CTE lesions and NFTs are densely distributed throughout the cerebral cortex, diencephalon, and brain stem with neuronal loss, gliosis, and astrocytic tau pathology. Chronic traumatic encephalopathy pathology in stages I and II is considered to be mild and in stages III and IV is considered to be severe.

Neuropathological evaluation was blinded to the clinical evaluation and was reviewed by 4 neuropathologists (V.A., B.H., T.D.S., and A.M.); any discrepancies in the neuropathological diagnosis were solved by discussion and consensus of the group. In addition to diagnoses, the density of tau immunoreactive NFTs, neurites, diffuse amyloid- β plaques, and neuritic amyloid- β plaques; vascular amyloid- β ; pTDP-43; and α -synuclein immunoreactive Lewy bodies were measured semiquantitatively (0-3, with 3 being most severe) across multiple brain regions.

Descriptive statistics were generated using SPSS software version 20 (IBM Inc).

Results

Among the 202 deceased brain donors (median age at death, 66 years [interquartile range [IQR], 47-76 years]), CTE was neuropathologically diagnosed in 177 (87%; median age at death, 67 years [IQR, 52-77 years]; mean years of football participation, 15.1 [SD, 5.2]; 140 [79%] self-identified as white and 35 [19%] self-identified as black), including 0 of 2 pre-high school, 3 of 14 high school (21%), 48 of 53 college (91%), 9 of 14 semiprofessional (64%), 7 of 8 Canadian Football League (88%), and 110 of 111 NFL (99%) players.

The median age at death for participants with mild CTE pathology (stages I and II) was 44 years (IQR, 29-64 years) and for participants with severe CTE pathology (stages III and IV) was 71 years (IQR, 64-79 years). The most common cause of death for participants with mild CTE pathology was suicide (12 [27%]) and for those with severe CTE pathology was neurodegenerative (ie, dementia-related and parkinsonian-related causes of death) (62 [47%]). The severity of CTE pathology was distributed across the highest level of play, with all former high school players having mild pathology (3 [100%]) and the majority of former college (27 [56%]), semiprofessional (5 [56%]), Canadian Football League (6 [86%]), and NFL (95 [86%]) players having severe pathology. The mean duration of play for participants with mild CTE pathology was 13 years (SD, 4.2 years) and for participants with severe CTE pathology was 15.8 years (SD, 5.3 years) ([Table 1](#)).

In all cases, perivascular clusters of ptau immunoreactive NFTs diagnostic for CTE (ie, CTE lesions)⁸ were found in the cerebral cortex ([Figure 1](#) and [Figure 2](#)). In cases with mild CTE pathology (stages I and II), isolated perivascular CTE lesions were found at the sulcal depths of the cerebral cortex, most commonly in the superior and dorsolateral frontal cortices, but also in the lateral temporal, inferior parietal, insula, and septal cortices ([Figure 1](#)). Neurofibrillary tangles were sparse in other cortical regions, and there was no diffuse neurofibrillary degeneration of the medial temporal lobe structures ([Figure 1](#), open arrowheads). Neurofibrillary tangles were also found in the locus coeruleus, substantia nigra, and substantia innominata ([Figure 3](#)) in mild CTE. In cases with severe CTE pathology, perivascular CTE lesions were large and confluent ([Figure 2](#)). Neurofibrillary tangles were widely distributed in the superficial laminae of cortical regions and there was severe neurofibrillary degeneration of the medial temporal lobe structures, including the hippocampus, amygdala, and entorhinal cortex ([Figure 2](#), black arrowheads, and [Figure 3](#)). Neurofibrillary tangles were also frequent in the thalamus, nucleus basalis of Meynert, substantia innominata, substantia nigra, and locus coeruleus in severe CTE ([Figure 3](#)).

Deposition of amyloid- β was present in a subset of participants at all stages of CTE pathology, predominantly as diffuse amyloid- β plaques, but neuritic amyloid- β plaques and amyloid angiopathy were also present. In stage IV CTE, amyloid- β deposition occurred in 52 cases (91%). Deposition of TDP-43 and α -synuclein were found in all stages of CTE pathology; TDP-43 deposition occurred in 47 (83%) and α -synuclein deposition occurred in 23 (40%) stage IV CTE cases ([Table 2](#)).

Among the 25 football players without CTE, 9 showed no pathological abnormalities and 7 showed nonspecific changes; eg, hemosiderin-laden macrophages (n=7) and axonal injury (n=5). Other diagnoses included vascular pathology (n=4), unspecified tauopathy not meeting criteria for CTE (n=3), AD (n=2), argyrophilic grain disease (n=1), and Lewy body disease (n=1).

Data on informants were collected beginning in 2014. The median number of participating informants was 2 (IQR, 1-3) per participant. Among all of the interviews,

71 (64%) included a spouse/partner, 56 (51%) included an adult child, 27 (24%) included a sibling, 16 (14%) included a parent, 13 (12%) included a non-first-degree relative, 8 (7.2%) included a neighbor or friend, and 4 included other informants. Among the informants who knew the participant the longest, the mean relationship length was 45.8 years (SD, 1.5 years).

Among the 111 CTE cases with standardized informant reports on clinical symptoms, a reported progressive clinical course was common in participants with both mild and severe CTE pathology, occurring in 23 (85%) mild cases and 84 (100%) severe cases (Table 3). Behavioral or mood symptoms were common in participants with both mild and severe CTE pathology, with symptoms occurring in 26 (96%) mild cases and 75 (89%) severe cases. Impulsivity, depressive symptoms, apathy, and anxiety occurred in 23 (89%), 18 (67%), 13 (50%), and 14 (52%) mild cases and 65 (80%), 46 (56%), 43 (52%), and 41 (50%) severe cases, respectively. Additionally, hopelessness, explosivity, being verbally violent, being physically violent, and suicidality (including ideation, attempts, or completions) occurred in 18 (69%), 18 (67%), 17 (63%), 14 (52%), and 15 (56%) mild cases, respectively. Substance use disorders were also common in participants with mild CTE, occurring in 18 (67%) mild cases. Symptoms of posttraumatic stress disorder were uncommon in both groups, occurring in 3 (11%) mild cases and 9 (11%) severe cases.

Cognitive symptoms were common in participants with both mild and severe CTE pathology, with symptoms occurring in 23 (85%) mild cases and 80 (95%) severe cases. Memory, executive function, and attention symptoms occurred in 19 (73%), 19 (73%), and 18 (69%) mild cases and 76 (92%), 67 (81%), and 67 (81%) severe cases, respectively. Additionally, language and visuospatial symptoms occurred in 54 (66%) and 44 (54%) severe cases, respectively. A premortem diagnosis of AD and a postmortem (but blinded to pathology) consensus diagnosis of dementia were common in severe cases, occurring in 21 (25%) and 71 (85%), respectively. There were no asymptomatic (ie, no mood/behavior or cognitive symptoms) CTE cases. Motor symptoms were common in severe cases, occurring in 63 (75%). Gait instability and slowness of movement occurred in 55 (66%) and 42 (50%) severe cases, respectively. Symptom frequencies remained similar when only pure CTE cases (ie, those with no neuropathological evidence of comorbid neurodegenerative disease) were considered (eTable in the Supplement).

Among the 111 CTE cases with standardized informant reports on clinical symptoms, 47 (42.3%; median age at death, 76 years [IQR, 63-81 years]) initially presented with cognitive symptoms, 48 (43.2%; median age at death, 66 years [IQR, 54-73 years]) initially presented with behavior or mood symptoms, and 16 (14.4%; median age at death, 65.5 years [IQR, 39-78]) initially presented with both cognitive symptoms and behavior or mood symptoms. Forty (85%) of those initially presenting with only cognitive symptoms were reported to have behavior or mood symptoms at the time of death and 43 (90%) of those initially presenting with only behavior or mood symptoms were reported to have cognitive symptoms at the time of death. Dementia was present

at the time of death in 36 (77%) of those initially presenting with cognitive symptoms, 33 (69%) of those initially presenting with behavior or mood symptoms, and 11 (69%) of those initially presenting with both cognitive and behavior or mood symptoms.

The most common primary cause of death was neurodegenerative for all 3 groups (cognitive, 26 [55%]; behavior or mood, 16 [33%]; both cognitive and behavior or mood, 6 [38%]). Substance use disorders, suicidality, and family history of psychiatric illness were common among those who initially presented with behavior or mood symptoms, occurring in 32 (67%), 22 (47%), and 23 (49%) cases, respectively.

Discussion

In a convenience sample of 202 deceased former players of American football who were part of a brain donation program, a high proportion were diagnosed neuropathologically with CTE. The severity of CTE pathology was distributed across the highest level of play, with all former high school players having mild pathology and the majority of former college, semiprofessional, and professional players having severe pathology. Behavior, mood, and cognitive symptoms were common among those with mild and severe CTE pathology and signs of dementia were common among those with severe CTE pathology.

Nearly all of the former NFL players in this study had CTE pathology, and this pathology was frequently severe. These findings suggest that CTE may be related to prior participation in football and that a high level of play may be related to substantial disease burden. Several other football-related factors may influence CTE risk and disease severity, including but not limited to age at first exposure to football, duration of play, player position, cumulative hits, and linear and rotational acceleration of hits. Recent work in living former football players has shown that age at first exposure may be related to impaired cognitive performance²⁹ and altered corpus callosum white matter³⁰ and that cumulative hits may be related to impairment on self-report and objective measures of cognition, mood, and behavior,³¹ although it is unclear if any of these outcomes are related to CTE pathology. Furthermore, it is unclear if symptomatic hits (concussions) are more important than asymptomatic hits resulting in subconcussive injury. As with other neurodegenerative diseases, age may be related to risk and pathological severity in CTE. It will be important for future studies to resolve how different measures of exposure to football and age influence the outcome.

In cases with severe CTE pathology, accumulations of amyloid- β , α -synuclein, and TDP-43 were common. These findings are consistent with previous studies that show deposition of multiple neurodegenerative proteins after exposure to TBI³² and with work showing that neuritic amyloid- β plaques are associated with increased CTE neuropathological stage.³³ Diagnoses of comorbid neurodegenerative diseases, including AD, Lewy body disease, motor neuron disease, and frontotemporal lobar degeneration, were also common in cases with severe CTE pathology. Overall, 19% of participants with CTE had comorbid Lewy body disease, which aligns with a recent

observation by Crane et al³⁴ regarding the increased prevalence of Lewy body pathology after single TBI. Chronic traumatic encephalopathy was not assessed in the analysis by Crane et al; to investigate the possibility of CTE after single TBI would require more extensive sampling of the depths of the cortical sulci with ptau immunostaining, as silver stains typically do not detect CTE pathology.

Behavioral, mood, and cognitive symptoms were common among participants with either mild or severe CTE pathology. In participants with severe CTE pathology, there was marked ptau pathology in brain regions that have been associated with symptoms frequently reported: impulsivity, depressive symptoms, apathy, anxiety, and explosivity (prefrontal cortex, amygdala, locus coeruleus); episodic memory symptoms (hippocampus and entorhinal and perirhinal cortices); and attention and executive function symptoms (prefrontal cortex). Participants with mild CTE pathology often had these symptoms despite having relatively circumscribed cortical pathology and absence of ptau pathology in the hippocampus, entorhinal cortex, or amygdala. This may suggest that other pathologies not captured by the pathological data set, such as neuroinflammation, axonal injury, or astrogliosis, or pathologies in neuroanatomical regions not evaluated contribute to these clinical symptoms. Microglial neuroinflammation appears to precede tau accumulation in CTE,³⁵ suggesting it may play a role in early symptoms.

Informants reported that 43% of participants had behavior or mood symptoms as their initial presentation. Many of these participants had a substance use disorder, demonstrated suicidality, or had a family history of psychiatric illness. Behavior or mood symptoms may be the initial presentation for a subset of individuals with CTE, or alternatively, CTE ptau pathology may lower the threshold for psychiatric manifestations in susceptible individuals. These clinical observations confirm and expand on previous reports of 2 primary clinical presentations of CTE.⁹

There is substantial evidence that CTE is a progressive, neurodegenerative disease. In this study, 107 participants (96%) had a progressive clinical course based on informant report. In addition, pathological severity of CTE was correlated with age at death (Table 3). However, a postmortem study evaluates brain pathology at only 1 time point and is by definition cross-sectional. In addition, the participants were not observed longitudinally during life. Although associations with age in cross-sectional samples can result from age-related progression within individuals, they can also arise from birth cohort effects, differential survival, or age-related differences in how individuals were selected into the study. Population-based prospective studies are needed to address the issue of progression of CTE pathology and age at symptom onset.

The strengths of this study are that this is the largest CTE case series ever described to our knowledge, more than doubling the size of the 2013 report,⁶ and that all participants were exposed to a relatively similar type of repetitive head trauma while playing the same sport. In addition, the comprehensive neuropathological evaluation

and retrospective clinical data collection were independently performed while blinded to the findings of the other investigators.

This study had several limitations. First, a major limitation is ascertainment bias associated with participation in this brain donation program. Although the criteria for participation were based on exposure to repetitive head trauma rather than on clinical signs of brain trauma, public awareness of a possible link between repetitive head trauma and CTE may have motivated players and their families with symptoms and signs of brain injury to participate in this research. Therefore, caution must be used in interpreting the high frequency of CTE in this sample, and estimates of prevalence cannot be concluded or implied from this sample. Second, the VA-BU-CLF brain bank is not representative of the overall population of former players of American football; most players of American football have played only on youth or high school teams, but the majority of the brain bank donors in this study played at the college or professional level. Additionally, selection into brain banks is associated with dementia status, depression status, marital status, age, sex, race, and education.³⁶ Third, this study lacked a comparison group that is representative of all individuals exposed to American football at the college or professional level, precluding estimation of the risk of participation in football and neuropathological outcomes.

Conclusions

In a convenience sample of deceased football players who donated their brains for research, a high proportion had neuropathological evidence of CTE, suggesting that CTE may be related to prior participation in football.

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Article Information

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Concept and design: Mez, Daneshvar, Abdolmohammadi, Murphy, Montenegro, Kowall, Cantu, Stern, McKee.

Acquisition, analysis, or interpretation of data: Mez, Daneshvar, Kiernan, Abdolmohammadi, Alvarez, Huber, Alosco, Solomon, Nowinski, McHale, Cormier,

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Supervision: Mez, Daneshvar, Abdolmohammadi, Solomon, Cantu, Stern, McKee.

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CUMULATIVE EXHIBIT B

MAF Physician Locator Tool

In this field, enter the street, city, state and zip code address of the Retired Player's primary residence to find the five closest Qualified MAF Physicians.

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A Qualified MAF Physician is a board-certified neurologist, board-certified neurosurgeon, or other board-certified neuro-specialist physician, who is part of an approved list of physicians authorized to make a Qualifying Diagnosis.

Contact an approved Qualified MAF Physician to schedule your appointment. You will be responsible for the costs of your exam. Where we have the information, we list the insurance plans accepted by each physician, but be sure to confirm whether your specific plan is accepted with the doctor's office before your appointment. The office of the Qualified MAF Physician who examines you must be located within 150 miles from your primary residence. Please note that this requirement applies only to appointments made after April 11, 2019. If you made your appointment before that date, you do not need to reschedule with a different Qualified MAF Physician. Use the mileage shown in the results from your search to pick a physician within 150 miles. Do not use the actual driving mileage to determine whether a Qualified MAF Physician meets this requirement. If there are no Qualified MAF Physicians in your search results within 150 miles, then contact us for help.

If the Retired Player wants to use a Qualified MAF Physician more than 150 miles away, we can make exceptions to that 150-Mile Rule but must do so before the appointment. This is a flexible rule with broad exceptions. We may grant exceptions to this 150-Mile Rule if the Retired Player asks us, where there is no Qualified MAF Physician close enough to him, or the wait for one is too long, or the Retired Player already has a doctor-patient relationship with a doctor, or other circumstances seem reasonable, such as, for example where the Retired Player has a secondary residence.

There are three ways to request approval to see a Qualified MAF Physician more than 150 miles from the Retired Player's primary residence: (1) click here to fill out and submit a request online; (2) click here to open a PDF of the request form you can print; or (3) email us at ClaimsAdministrator@NFLConcussionSettlement.com and tell us the information requested in the form. We will let you know as quickly as possible whether your request has been approved.

If you have any questions about any of this or have any problems getting an appointment, email us at ClaimsAdministrator@NFLConcussionSettlement.com or call us at 1-855-887-3485.

MAF Physician Locator Search Results

1. Jaroslaw Koberda, MD
J. Lucas Koberda, MD, PhD,
Neurology, PL

4838 Kerry Forest Parkway
 Tallahassee, FL 32309
 850-877-2802

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2. Michael Scott Jaffee, MD
University of Florida
Department of Neurology

1149 Newell Drive
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 352-273-8500

Approx. 127 miles
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Some of these Qualified MAF Physicians are identified as "Pending." This means the physician has been approved as a Qualified MAF Physician, but there are still some steps required to complete the paperwork. You can contact these physicians to schedule the appointment as we expect these final steps to be completed very soon.

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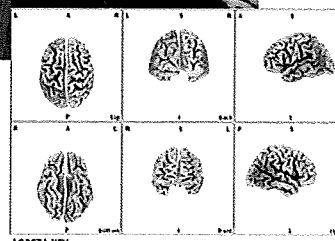
AREAS OF EXPERTISE:

Alzheimer's disease (AD) is a neurodegenerative illness affecting (with other dementias) approximately 500,000 Floridian's causing progressive forgetfulness and other cognitive dysfunctions. It is suspected that it is caused by the accumulation in a brain of abnormal protein called amyloid. It is estimated that the deposition of the amyloid in a brain starts 5-10 years before the first symptoms of memory problems are noticed by the patient or his/her family. Recently, the amyloid detection nuclear imaging has been approved by the FDA for use in clinical settings.

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Our Mission:

- Provide patient care that is helpful to all, including those patients who do not respond to multiple medications.
- Improve brain function in patients with traumatic brain injury (TBI), memory problems, epilepsy, anxiety, depression, attention deficit disorder (ADD/ADHD), and autism spectrum disorder.
- Offer care that is prompt and cost effective.
- Perform research in the area of cognition/brain function improvement.
- Peak Performance training for academic and professional athletes--anxiety reduction, enhance focus and coordination

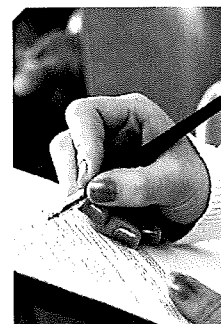
Our Services:

- Neurological Consultation
- Electroencephalography (EEG)
- Neuro Cognitive Testing
- Neurofeedback

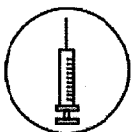


New Patient?

[Download
our
registration
forms](#)



[Where To Find Us >](#)



Tallahassee Local?

Call to hear about our Neurofeedback package deal (850) 877-2802

Dr. J Lucas Koberda M.D. Ph.D.
4838 Kerry Forest Pkwy
Tallahassee, FL 32309



**For Life-Threatening
Emergencies Call 911**

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Dr. Koberda M.D. Ph.D.
Neurology and Brain Enhancement



4838 Kerry Forest Pkwy
Tallahassee, FL 32309



(850) 877-2802
FAX: (850) 222-1383

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Testimonials

We love to hear from our patients! Click the button below to write about your experience at our clinic.

*Disclaimer: statements made by any person(s) within this page are not intended to substitute medical evaluation and are not to be seen as a guarantee of outcomes.

[Submit Testimonial](#)

Renee 10/02/2018

Dr. Koberda is wonderfully kind and patient. He is welcoming. I am an African-American woman who is recovering from a concussion after a terrible accident. Dr. Koberda made me feel welcomed on every single visit. Since my concussion, I had constant headaches, speech trouble, memory problems, problems focusing, and light sensitivity. A previous doctor had only prescribed me medicine, however, Dr. Koberda offered me much more! I gained a chance to understand more about what my brain was undergoing and ways that I could care for myself. I underwent neural therapy options in his office, and my stomach was happy that medicines were not upon it! I feel so much better now! Recovery is possible and it can be facilitated with Dr. Koberda's specialized expertise. I would encourage anyone of any background to visit his office for his neural care.

MSW 08/19/2016

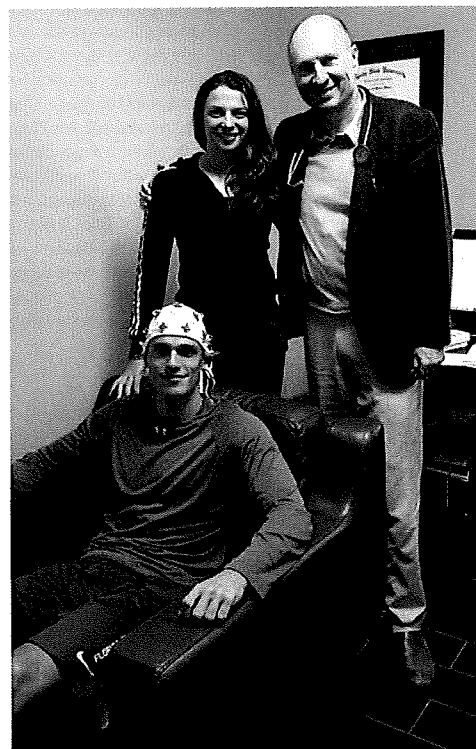
I was diagnosed with Lyme last February. Classic symptoms of Lyme include insomnia and fractionated sleep, which inhibits deep healing. I have recently completed 10 sessions of neurofeedback training for sleep problems, and have to say I am extremely happy with the results. Before I began the training, I had not been dreaming for months. By the 10th session, I was having vivid dreams again, and remembering them. I bought a Jawbone Up before the training in order to measure the amount of time each night I was in REM, light, and deep sleep. Dr. Koberda mentioned that we all need at least 3 hours a night of deep plus REM sleep in order to feel rested. By the end of the training, I was sleeping through the night and my combined REM plus deep sleep time rose from around 2.5 hours per night to 4.5 to 5 hours a night. I feel rested in the mornings now, have no trouble staying asleep at night, have more stable moods, and basically feel mentally and physically great. I can also concentrate better and for longer periods of time at work. This is a wonderful program for anyone with sleep problems, anxiety, and depression. Dr. Koberda is a world-class physician, and Tallahassee is extremely fortunate to have someone of his caliber practicing his specialty here. Thank you, Dr. Koberda!

George 05/26/2015

I was having issues relating to retirement and boredom. I had been seeing a stress counselor, who recommended Dr. Koberda. Being a lawyer, I was somewhat skeptical that these neurofeedback sessions would help. They did, and after completion of these sessions, I felt better about myself and my life in general. The positiveness that I have always prided myself on, fully returned. I no longer need the medication that was prescribed by others for me. I understand that this type of treatment is cutting edge; I certainly hope more of this type of treatments are made available and covered by insurance. I am so thankful that I was able to overcome the circumstances that led me to Dr. Koberda. Thank you, Dr. Koberda, and thanks to his staff.

Tamela 05/25/2015

I was in the midst of deep depression. seriously considering ECT (shock therapy)

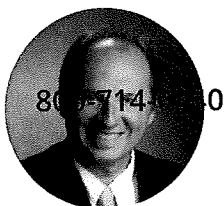




PURE RECOVERY
CALIFORNIA

Addiction Treatment Dual Diagnosis About Our Programs Media + News

Contact



LUCAS KOBERDA, MD, PHD, CONSULTANT

Dr. J. Lucas Koberda is a board-certified neurologist and an internationally trained physician who completed his residency in Neurology at the Oregon Health Sciences University in Portland, Oregon.

Prior to his neurological training, he received his Ph.D. based on his research in the area of tumor immunology. Dr. Koberda is currently affiliated with The Florida State University College of Medicine.

His main interest is in neuro-psychiatry and cognitive enhancement. He uses the newest technology of QEEG and Neurofeedback to successfully diagnose and treat many medical conditions including seizures, headaches, fibromyalgia chronic pain, anxiety, depression, and prior stroke.

Dr. Koberda has also effectively introduced neurofeedback protocols for cognitive enhancement for professionals to improve their memory, concentration, verbal function and information processing speed.

He was appointed in August 2010 by Governor Charlie Christ as a member of the Alzheimer's Disease Advisory Committee for a 4

year term (The Committee serves as an advising body to the Florida State Government).

Dr. Koberda has published multiple publications in different scientific journals and serves as an international neuroscience speaker and consultant. Dr. Koberda has been appointed to the Editorial Board of the "Journal of Neurology and Stroke," "International Journal of Emergency Mental Health and Human Resilience," "Journal of Neurology and Neurobiology," and "Journal of Psychology and Clinical Psychiatry."

He also served as a reviewer of "Clinical EEG and Neuroscience" and the above journals.

Read more about Pure Recovery Staff and Consultants or learn about our Executive Treatment Program in California.

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TREATMENT PROGRAM


- Our Services
- Detox
- MEDIA + NEWS
- Press
- Articles + Research
- BLOG
- TESTIMONIALS
- Clients
- Experts
- LEGAL

LOCATION

- Pure Recovery
California
- Corporate Office
4310 Tradewinds
Drive, Suite 300
Channel Islands, CA
93035
- Phone: (805) 815-3399

CONTACT US

To Speak With A
Specialist

 (800) 714-0340



info@purerecoveryca.com

JOINT COMMISSION



Dr. Koberda M.D. Ph.D.
Neurology and Brain Enhancement



4838 Kerry Forest Pkwy
Tallahassee, FL 32309



(850) 877-2802
FAX: (850) 222-1383

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Publications

Neurofeedback Training for Epilepsy--Lu...

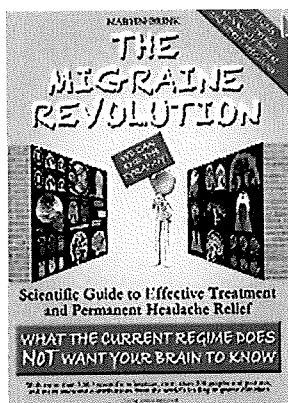


Dr. Koberda has been published in over 20 medical journals.

- [Neurofeedback and Alzheimer's](#)
- [Neurofeedback and Cognitive Enhancement](#)
- [Neurofeedback in General Neurology](#)
- [Neurofeedback and Dementia](#)
- [Neurofeedback and Stroke](#)
- [Neurofeedback and ADD/ADHD](#)
- [Neurofeedback and Epilepsy](#)
- [Neurofeedback and Epilepsy 2](#)
- [Neurofeedback and Epilepsy Poster](#)
- [Neurofeedback and Mental Disorders](#)
- [Neurofeedback and TBI](#)
- [Neurofeedback and Pain Management](#)
- [Neurofeedback and Headaches](#)
- [Neurofeedback for Depression/Anxiety](#)
- [Masgutova Neurosensorimotor Reflex Integration \(MNRI\) Neuromodulation Technique](#)
- [Neuromodulation is an Emerging Modality](#)
- [SmartDrugSmarts Neurofeedback Podcast](#)

Book Contributions

Dr. Koberda has written chapters for many books about neurology and neurofeedback.



THE MIGRAINE REVOLUTION: We can End the Tyranny! Scientific Guide to Effective Treatment and Permanent Headache Relief (What the Current Regime Does Not Want Your Brain to Know)

by Martin Brink

Find it on Amazon [HERE](#)
More Information [HERE](#)

Clinical Neurotherapy: Application of

Recommended Articles

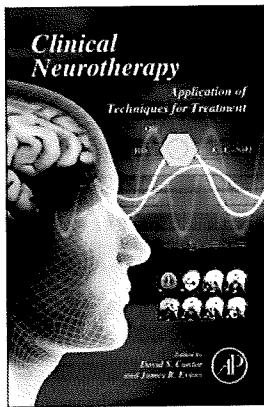
- [Neurofeedback Induces Changes in White + Gray Matter](#)
- [Results of 12-week Brain Fitness Program](#)
- [Neurofeedback and PTSD Plastic Modulation](#)
- [Neurofeedback in ADHD vs Medication](#)
- [Neurofeedback as Treatment for ADD/ADHD](#)
- [QEEG can discern reason for cognitive disorder](#)

Braintraining for Anxiety & ADHD



More Videos

- [Basketball Player Chris Kaman Uses Neurofeedback](#)
- [BBC: Treating Depression with Electromagnetic Therapy](#)
- [Neurobalancing with Dr. Koberda on Recovery Now](#)
- [Neurofeedback Therapy for Ft. Campbell Soldiers with TBI](#)

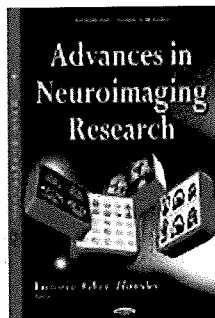


Clinical Neurotherapy. Application of Techniques for Treatment. 1st Edition.

Edited by David S. Cantor and James R. Evans

"Defining Developing Evidence Based Methods (EBM) databases proving treatment efficiency" - Dr. J. Lucas Koberda

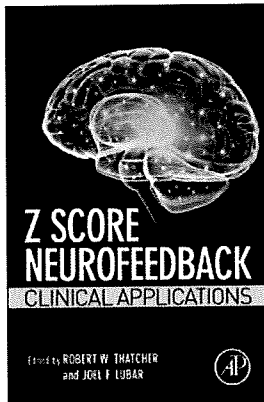
Find it on Amazon [HERE](#)



Advances in Neuroimaging Research

Edited by Victoria Ashler-Hansley

"LORETA imaging and LORETA neurotherapy in neuropsychiatry" - Dr. J. Lucas Koberda



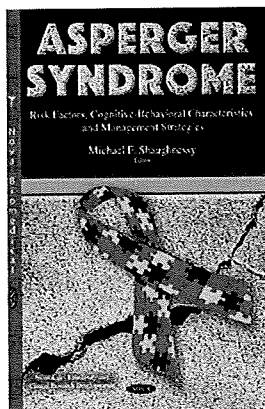
Z-Score Neurofeedback: Clinical Applications.

Edited by Robert W. Thatcher and Joel F. Lubar

Chapter 5-"Z-score LORETA neurofeedback as a Potential Therapy in Depression/Anxiety and Cognitive Dysfunction".

Chapter 6-"LORETA Z-score Neurofeedback in Chronic Pain and Headaches".

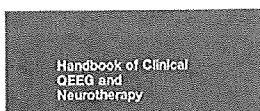
Chapter 10-"Therapy of Seizures and Epilepsy with Z-score LORETA Neurofeedback" - Dr. J. Lucas Koberda



Asperger Syndrome: Risk Factors, Cognitive Behavioral Characteristics, and Management Strategies.

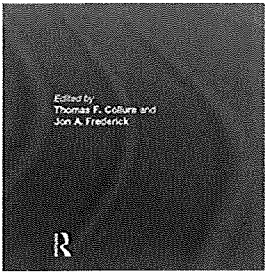
Edited by Michael F. Shaughnessy

"QEEG/LORETA Electrical Imaging and Z-score Neurofeedback: New Approach to Diagnosis and Therapy of Autistic Spectrum Disorders (ASD)" - Dr. J. Lucas Koberda



Handbook of Clinical QEEG and Neurotherapy

Edited by Thomas F. Collura and Jon A. Koberda



Frederick
"LORETA Z-score Neurofeedback in the
Neuropsychiatric Practice"" - Dr. J. Lucas
Koberda

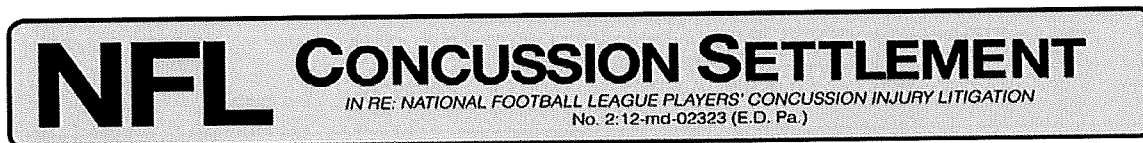
Dr. J Lucas Koberda M.D. Ph.D.
4838 Kerry Forest Pkwy
Tallahassee, FL 32309



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Emergencies Call 911**

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© 2018 Dr. J Lucas Koberda M.D. Ph.D. Neurology



NETWORK PROVIDER APPLICATION

Please complete and return this application with the following supporting documents:

- A copy of your state license to operate;
- Proof of Medicare and/or Medicaid certification;
- Certificate(s) of Insurance proving current general liability and professional liability/medical malpractice insurance coverage and amounts of coverage;
- A copy of your organization's W-9 form (August 2013 or later); and
- If you or any of the facilities or practice sites in your system participate in one or more state-sponsored or state-affiliated patient compensation funds, please include (a) the name of the fund(s), (b) a list of your service or practice sites participating in each such fund, (c) your certificate(s) of coverage, and (d) the declaration page(s) for your underlying primary coverage(s), general and professional liability.

This application does not constitute a contract.

Please furnish the information below. Indicate "N/A" if an item is not applicable.

ORGANIZATION INFORMATION**I. GENERAL INFORMATION**

Please provide the following general information about the your organization:

J. Lucas Koberda, MD, PhD, Neurology, PL

Organization Name

ME88946

01/01/2018

State License Number

Expiration Date

1 6 - 1 5 5 1 2 0 2

1972548279

Tax ID

NPI

4838 Kerry Forest Parkway

Street Address

Tallahassee

FL

32309

City

State

ZIP

J. Lucas Koberda

Doctor/Owner

Primary Contact Name

Title

(850) 877-2802

jlkoberda@yahoo.com

Primary Contact Phone

Primary Contact E-mail

Parent company or organization (if applicable): N/A

II. LIABILITY COVERAGE**A. Does your organization maintain the following types of insurance coverage?**

Yes No N/A

1. Commercial general liability for bodily injury/property damage and contractual liability

☒ ☐ ☐

If so, in what amounts? \$1,000,000 (occurrence) \$2,000,000 (aggregate)

2. Professional liability and/or medical malpractice insurance

☒ ☐ ☐

If so, in what amounts? \$250,000 (occurrence) \$750,000 (aggregate)

B. Has your organization experienced any of the following:

Yes No N/A

1. Malpractice liability insurance cancellation in the past five (5) years?

☐ ☒ ☐

2. General liability insurance cancellation in the past five (5) years?

☐ ☒ ☐

3. Revocations or suspensions as a Medicare or Medicaid Provider?

☐ ☒ ☐

4. State licensing investigations or actions?

☐ ☒ ☐

If you answered "Yes" to any question(s) in Section B on the preceding page, please provide an explanation below, as well as any additional relevant information about your organization:

III. DELIVERY SITES

Please provide the following information for each site your organization proposes to provide services in connection with the NFL Concussion Settlement. Please include a separate entry for each proposed delivery site:

J. Lucas Koberda, MD., PhD., Neurology, PL.- AKA Tallahassee NeuroBalance Center		
Name of Facility		
4838 Kerry Forest Parkway		
Street Address		
Tallahassee	FL	32309
City	State	ZIP
(850) 877-2802	(850) 877-2802	(850) 222-1383
Primary Telephone Number	Scheduling Telephone Number	Fax Number
ME88946	1972548279	
State License Number	NPI Number	

Please see Application Instructions on page 1 for documentation requirements.

IV. PRACTITIONERS

Please provide the following information for each practitioner that your organization proposes to provide services covered under the NFL Concussion Settlement. Please include a separate entry for each proposed practitioner.

Qualified BAP Providers must be one of the following:

- A clinical neuropsychologist certified by the ABPP or ABCN in the specialty of Clinical Neuropsychology; or
- A board-certified neurologist.

Qualified MAF Physicians must be one of the following:

- A board-certified neurologist;
- A board-certified neurosurgeon; or
- A board-certified neuro-specialist physician.

All practitioners seeking to serve as a Qualified BAP Provider or Qualified MAF Physician must meet the following requirements:

- Possess a current, active, unrestricted state license;
- Hospital staff privileges not revoked or restricted within the past five (5) years;
- Is covered by proper insurance under state law;
- Not excluded from participation in any federal or state health care program; and
- Medical license not subjected to any disciplinary action or any restrictions within the past five (5) years.

Koberda	Jaroslav	L
Last Name	First Name	MI
MD, PhD	ME88946	
Professional Designation (e.g., M.D., Ph.D.)	State License Number	
Gdansk Medical University (Medical School)-Poland		
Education		
Oregon Health Sciences University, Portland OR-Neurology Residency Training, USA		
Training		
Extensive experience including clinical work and publications		
Experience with Sports-Related Concussions or Traumatic Brain Injury		
1927548279	Neurology	
NPI	Specialty	
Yes-The American Board of Psychiatry and Neurology		
Board Certification		
19	40	English, Polish
No. of Years in Practice as a Healthcare Provider	Hours per Month Available To See Settlement Participants	Languages Spoken
4838 Kerry Forest Parkway Tallahassee, FL 32309		
Location(s) Where the Practitioner Provides Services		

Average wait time for new patient evaluations (number of days from request for appointment until the patient can be seen by the practitioner): 1 week

Is the practitioner an employee of your organization? Yes ☒ No ☐

Please see Application Instructions on page 1 for documentation requirements.

If you answered "No" to the preceding question, please describe the relationship between the practitioner and the organization and name the practitioner's employer:

Self employed-J. Lucas Koberda, MD., PhD., Neurology, PL

LIABILITY COVERAGE

A. Does the practitioner carry these types of insurance coverage?

Professional liability and/or medical malpractice insurance

Yes No N/A

☒ ☐ ☐

If No, is the practitioner covered by your organization's liability and/or malpractice insurance?

☐ ☐ ☐

If Yes, please indicate the following:

Type of insurance: _____ Amounts: \$250,000 (occurrence) \$750,000 (aggregate)

Type of insurance: _____ Amounts: \$ _____ (occurrence) \$ _____ (aggregate)

Type of insurance: _____ Amounts: \$ _____ (occurrence) \$ _____ (aggregate)

B. Has the practitioner experienced any of the following:

Yes No N/A

1. Malpractice liability insurance cancellation?

☐ ☒ ☐

2. General liability insurance cancellation?

☐ ☒ ☐

3. Cancellation of any other insurance policies related to the practice of medicine?

☐ ☒ ☐

4. Revocations or restrictions of hospital staff privileges?

☐ ☒ ☐

5. Revocations or suspensions as a Medicare or Medicaid Provider?

☐ ☒ ☐

6. State licensing investigations or actions?

☐ ☒ ☐

If you answered "Yes" to any of the preceding questions, please provide details and dates below:

Percentage of practice related to litigation (expert/consulting engagements) for plaintiffs, defendants and court/administrative bodies, and a description of such practice since July 1, 2011:

Plaintiffs: $\frac{7}{\text{Percentage}}$ See attached

Defendants: $\frac{4}{\text{Percentage}}$ See attached

Court/Admin: $\frac{0}{\text{Percentage}}$

Please see Application Instructions on page 1 for documentation requirements.

List all engagements as a litigation expert consultant or expert witness arising out of, or relating to, head, brain and/or cognitive injury. Please include any and all engagements (irrespective of date) and list the subject matter, client, and date range. If the engagement included testimony (including, but not limited to, the preparation of an expert report), provide the title, docket number and court of the proceeding:

Over the 19 years of my neurology practice I have testified in 8 workers' compensation cases (4 times for the defense and 4 times for the plaintiff); 2 social security cases for the plaintiff; 1 disability benefits case for the plaintiff; and 1 death penalty case for the state. Details on title, docket number and court of the proceeding are attached.

Has the practitioner served on or after April 22, 2015 as a litigation expert consultant or expert witness for an Opt Out of the NFL Concussion Settlement, in connection with litigation relating to head, brain and/or cognitive injury? If Yes, please provide details below:

Yes ☐ No ☒

Has the practitioner ever treated or evaluated a current or former NFL player? If Yes, please provide a general description below (without identifying the player(s)):

Yes ☒ No ☐

Over 19 years of my neurology practice I have seen former NFL players in hospital settings (psychiatric hospital) and in my office as regular patients, clinical research, and for IME evaluations.

Has the practitioner ever been in a salaried position or consulting relationship with the National Football League, NFL Properties, or any NFL Member Clubs? If Yes, please provide details below, including the title of the practitioner's position/role:

Yes ☐ No ☒

Has the practitioner ever served as a neutral physician or consultant for benefits under the NFL Player Disability & Neurocognitive Benefit plan, the Bert Bell/Pete Rozelle NFL Retirement Plan or the 88 Plan? If Yes, please provide details below, including the title of practitioner's position/role:

Yes ☐ No ☒

Has the practitioner ever been convicted of a crime of dishonesty? If Yes, please describe the crime and date of conviction:

Yes ☐ No ☒

Please also attach a current curriculum vitae for each proposed practitioner.

Please see Application Instructions on page 1 for documentation requirements.

V. BILLING/CLAIMS PROCESS

Please note: This information pertains only to the Baseline Assessment Program. All services provided by Qualified MAF Physicians are the responsibility of the Retired NFL Football Player and/or his insurer.

What is your preferred billing process?

- ☒ Electronic claim submission ☐ Fax
☐ Upload to a secure portal ☐ Mail (e.g., CMS1500, UB04)

Does your system contract with a third-party company to manage billing? Yes ☐ No ☒

If "Yes," please provide the following information:

Billing Company Name

Street Address 1

Street Address 2

City

County

State

ZIP

Billing Contact Name

Phone

Is your billing address different from your mailing address? If so, please provide the billing address:

Name

Street Address 1

Street Address 2

City

County

State

ZIP

Phone

Fax

Contact Person

Title

Phone

E-mail

Please see Application Instructions on page 1 for documentation requirements.

VI. PAYMENT

Please note: This information pertains only to the Baseline Assessment Program. All services provided by Qualified MAF Physicians are the responsibility of the Retired NFL Football Player and/or his insurer.

Do you prefer to be paid by check or ACH deposit?

☒ Check ☐ ACH Deposit

Provide the mailing address to which Explanations of Payment should be mailed:

4838 Kerry Forest Parkway

Street Address 1

Street Address 2

Tallahassee

City

Leon

County

FL

State

32309

ZIP

Provide the mailing address to which 1099 Statements should be mailed:

4838 Kerry Forest Parkway

Street Address 1

Street Address 2

Tallahassee

City

Leon

County

FL

State

32309

ZIP

VII. INSURANCE PLANS ACCEPTED

Please note: This information pertains only to the those applying for participation as a Qualified MAF Physician. Anyone applying for participation as a Qualified BAP Provider only may omit this information.

List the insurance plans accepted by your organization:

Medicare, BC/BS, United Healthcare-PPO, Capital Health Care

Please see Application Instructions on page 1 for documentation requirements.

DECLARATION

The undersigned attests that he or she has the authority to act on behalf of the applicant for the purpose of this application. The applicant, by and through the undersigned, attests that to the best of its knowledge and belief, after reasonable inquiry, all of the information provided on this application and in connection with this application is complete and accurate. The applicant understands that this application does not entitle the applicant to participate in any program or work arising out of the NFL Concussion Settlement activities or any other program. The applicant further understands that any misrepresentations made in this application shall be grounds for immediate disqualification from participation in the programs arising out of the NFL Concussion Settlement. The applicant agrees that entities that in good faith provide information to Garretson Resolution Group (GRG) and/or BrownGreer to assist GRG and/or BrownGreer in evaluating and/or verifying the information contained in this application and in any documentation submitted in support of this application shall not be liable for any act or omission related to the provision, evaluation, or verification of such information. The applicant further agrees to notify GRG and/or BrownGreer in a timely manner of any changes to the information provided on this application.

The applicant hereby authorizes any accrediting body, governmental entity, insurance company, association, organization, entity, or person to release the information requested herein and to provide confirmation of the answers contained herein to GRG and/or BrownGreer and their affiliates, subsidiaries, and agents. This authorization shall be valid until and unless the applicant withdraws its application. A copy of the signature is as binding as the original.

Pursuant to 28 U.S.C. § 1746, I declare under penalty of perjury that the information provided in this form, and in any attachments hereto, is true and correct to the best of my knowledge, information, and belief.

Authorized Signature		Date	
Jaroslav Lucas Koberda			
Print Name			
J. Lucas Koberda, MD, PhD, Neurology PL, Tallahassee NeuroBalance Center			
Organization Name			
4838 Kerry Forest Parkway			
Street Address 1			
Street Address 2			
Tallahassee	Leon	FL	32309
City	County	State	ZIP

Please see Application Instructions on page 1 for documentation requirements.

EXHIBIT C

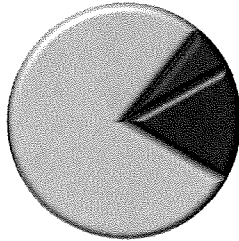
NFL CONCUSSION SETTLEMENT

IN RE: NATIONAL FOOTBALL LEAGUE PLAYERS' CONCUSSION INJURY LITIGATION
No. 2:12-md-02323 (E.D. Pa.)

NFL SETTLEMENT PROGRAM SUMMARY REPORT (As of 10/14/19)

SECTION 1: TIMELY REGISTRATIONS

TOTAL: 20,548 (17,243 RETIRED PLAYER AND REPRESENTATIVE CLAIMANTS)



■ Retired Player
15,919 (78%)

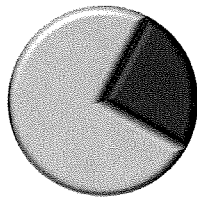
■ Representative Claimant
1,324 (6%)

■ Derivative Claimant
3,305 (16%)

SECTION 2: REGISTRATION NOTICES ISSUED BY TYPE TOTAL: 20,548

	15,000	12,836	6,554	1,158	0
	10,000				
	5,000				
	0				
		BAP Eligible	Not BAP Eligible	Adverse Notice	Incomplete Registration
TOTAL		12,836	6,554	1,158	0
■ Retired Player		12,691	2,426	802	0
■ Representative		145	876	303	0
■ Derivative		0	3,252	53	0

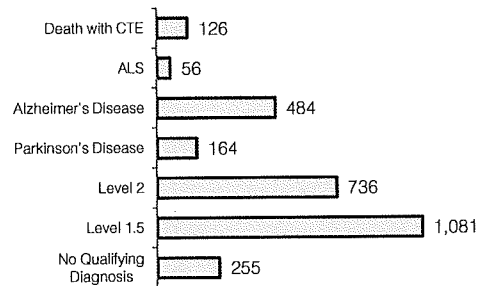
SECTION 3: MONETARY AWARD CLAIMS SUBMITTED: 2,902



■ Retired Player
2,161 (74%)

■ Representative Claimant
741 (26%)

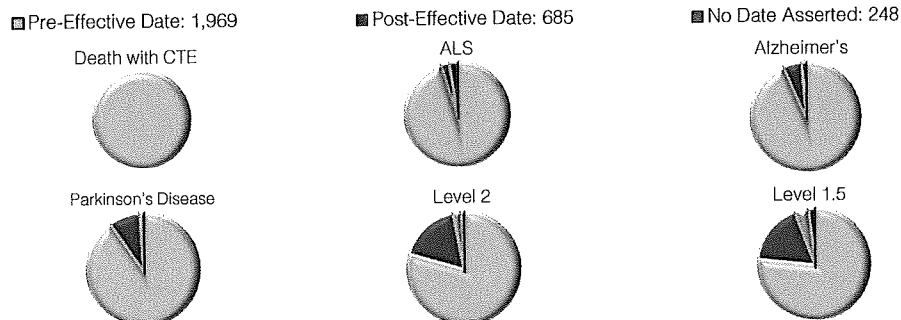
SECTION 4: QUALIFYING DIAGNOSIS ASSERTED IN MONETARY AWARD CLAIMS



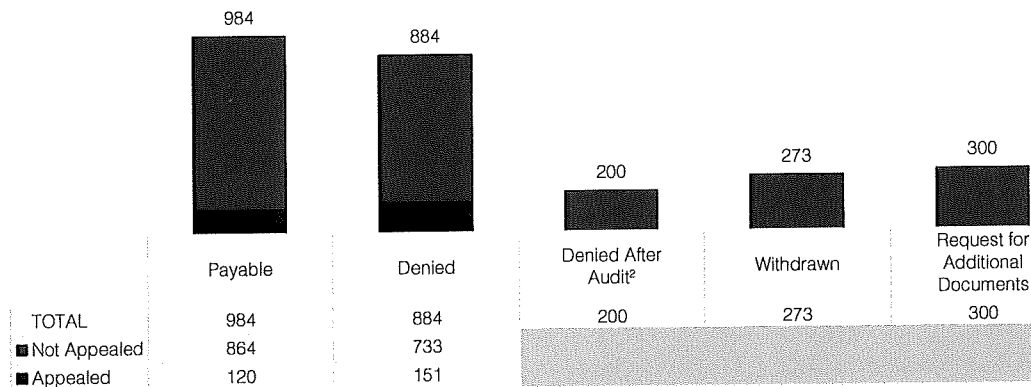
NFL CONCUSSION SETTLEMENT

IN RE: NATIONAL FOOTBALL LEAGUE PLAYERS' CONCUSSION INJURY LITIGATION
No. 2:12-md-02323 (E.D. Pa.)

SECTION 5: QUALIFYING DIAGNOSIS DATE ASSERTED IN MONETARY AWARD CLAIMS



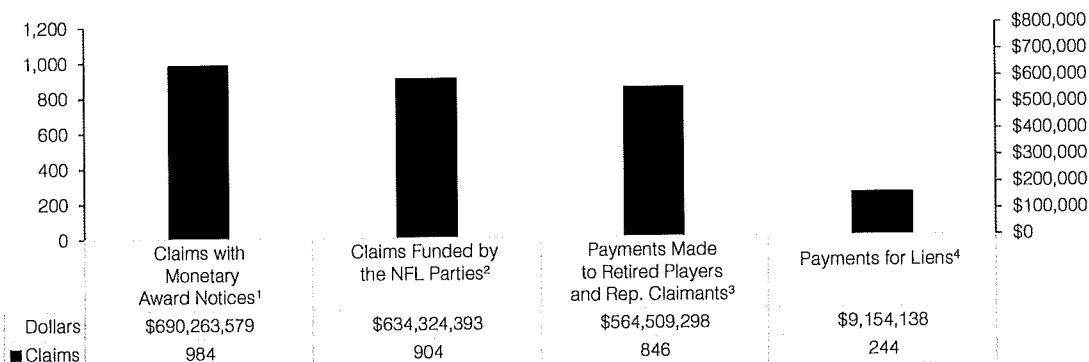
SECTION 6: NOTICES ISSUED ON MONETARY AWARD CLAIMS: 2,641¹



¹ Section 6 counts all Monetary Award claims that have received a notice after claim processing. Claims receiving multiple notices are counted only once based on the most recent notice issued. The number of appealed notices reflects only claims that have been appealed on the most recent notice issued. For a cumulative total of all appeals, see Section 9.

² The results of the audit closed these claims and directed further alternatives for these Settlement Class Members.

SECTION 7: PAYABLE CLAIMS SUMMARY



¹ The dollars and number of claims shown are the number of notices issued before holdbacks for potential Derivative Claimants, common benefit fees, liens and any determinations on appeals.

² The dollars and number of claims shown include claims from Retired Players and Representative Claimants.

³ The dollars shown include payments issued on behalf of Settlement Class Members to Third-Party Funders who have accepted rescission of prohibited assignments entered into with Settlement Class Members.

⁴ The claims shown reflect the Settlement Class Members on whose behalf the Settlement Program has issued payments to resolve liens. The dollars shown reflect all payments the Settlement Program has made on behalf of Settlement Class Members to resolve liens, including (1) payments to the Lien Resolution Administrator to resolve medical liens, (2) payments to the Lien Resolution Administrator for its lien fees, and (3) payments directly to lienholders to resolve non-medical liens.

NFL CONCUSSION SETTLEMENT <small>IN RE: NATIONAL FOOTBALL LEAGUE PLAYERS' CONCUSSION INJURY LITIGATION No. 2:12-md-02323 (E.D. Pa.)</small>										
SECTION 8 STATUS OF MONETARY AWARD CLAIMS (BASED ON LAST NOTICE OR ACTION ON CLAIM)										
		CTE	ALS	Alzheimer's Disease	Parkinson's Disease	Level 2	Level 1.5	No Qualifying Diagnosis	Total	%
1.	In Review Process at Claims Administrator	0	0	1	0	8	4	7	20	<1%
2.	MAF Rule 20 Deviation Explanation Required	0	0	0	0	18	29	0	47	2%
3.	Notice Ready to Issue	3	0	13	2	51	51	6	126	4%
4.	Needs Special Master Statute of Limitations Review	3	14	26	13	5	11	0	72	2%
5.	Last Notice was for Incomplete Claim Package ¹	0	0	9	0	15	22	7	53	2%
6.	Ready for Review by AAP	0	0	6	1	16	25	3	51	2%
7.	Last Notice was Award Notice ²	0	0	6	2	6	11	0	25	<1%
8.	Last Notice was Denial Notice ³	2	0	3	0	20	29	7	61	2%
9.	On Appeal Now ⁴	1	0	3	0	16	18	2	40	1%
10.	In Audit Investigation ⁵	0	0	3	1	12	16	0	32	1%
11.	In Audit-Failure to Provide Information	0	0	0	0	0	0	0	0	0%
12.	Referred to the Special Investigator by the Special Masters	0	0	2	0	74	99	1	176	6%
13.	Adverse Audit Report with the Parties for Review	0	0	0	0	0	0	0	0	0%
14.	Adverse Audit Report Awaiting Response from Those Audited	0	0	0	0	0	1	0	1	<1%
15.	Adverse Audit Report with the Special Masters for Review	0	0	0	0	1	0	0	1	<1%
16.	Denied After Audit ⁶	0	0	34	0	79	86	1	200	7%
17.	Final Denial	38	3	68	11	151	317	202	790	27%
18.	Withdrawn	0	0	15	3	129	107	19	273	9%
19.	Ready for Next Month's Payment List	0	0	0	0	1	0	0	1	<1%
20.	On this Month's Payment List	2	2	21	9	17	36	0	87	3%
21.	Paid ⁷	77	37	274	122	117	219	0	846	29%
22.	Total Claim Packages Submitted	126	56	484	164	736	1,081	255	2,902	100%

1. The Program has issued notices to 1,938 incomplete claim packages, which is 71% of all Level 1.5 and Level 2 claims and 60% of all other claims.

2. For details on all claims receiving an Award Notice and the dollar values assigned, see Sections 7 and 11.

3. For details on all denied claims, including the denial reasons, see Section 10.

4. For details on all appeals filed by Settlement Class Members and the NFL parties, see Section 9.

5. The Program has audited 1,233, or 43%, of all claims, including 775 claims removed from audit and put back into the claims review process, 208 claims currently in Audit Investigation, 2 claims in Adverse Audit Reports with the Parties or Special Masters for Review or awaiting a response from those audited, 48 claims that were withdrawn while in audit, and 200 claims denied after audit.

6. The results of the audit denied these claims and directed further alternatives for these Settlement Class Members.

7. For total dollars paid to or on behalf of these Settlement Class Members, see Part 5 of Section 11.

NFL**CONCUSSION SETTLEMENT**IN RE: NATIONAL FOOTBALL LEAGUE PLAYERS' CONCUSSION INJURY LITIGATION
No. 2:12-md-02323 (E.D. Pa.)**SECTION 9****STATUS OF ALL APPEALS ON MONETARY AWARD CLAIMS**

	Status ¹	Appealed by Class Member	Appealed by NFL Parties	Total
A.	Payable Claims	53	131	184
1.	Appeal Filed and in Preliminary Steps	1	0	1
2.	Appellee's Opposition Memo Not Received	0	4	4
3.	Appellee's Opposition Memo Received	0	3	3
4.	Remanded to Claims Administrator	21	5	26
5.	With Special Masters for Decision	3	7	10
6.	Result Upheld on Appeal	21	82	103
7.	Result Overturned on Appeal	3	15	18
8.	Closed or Withdrawn ²	4	15	19
B.	Denied Claims	258	0	258
1.	Appeal Filed and in Preliminary Steps	11	0	11
2.	Appellee's Opposition Memo Not Received	7	0	7
3.	Appellee's Opposition Memo Received	3	0	3
4.	Remanded to Claims Administrator	89	0	89
5.	With Special Masters for Decision	7	0	7
6.	Result Upheld on Appeal	106	0	106
7.	Result Overturned on Appeal	7	0	7
8.	Closed or Withdrawn ²	28	0	28
C.	TOTAL APPEALS	311	131	442

¹ Class Counsel has filed statements in 37 of the Appeals across multiple statuses in the Appeals Process.² These are appeals we are no longer processing because (1) the appealing party withdrew or (2) it was closed after being in audit.

NFL CONCUSSION SETTLEMENT <small>IN RE: NATIONAL FOOTBALL LEAGUE PLAYERS' CONCUSSION INJURY LITIGATION No. 2:12-md-02323 (E.D. Pa.)</small>		
SECTION 10 REASONS IN NOTICES OF DENIAL ISSUED ON MONETARY AWARD CLAIMS		
	Reason Claim Denied	Notices
1.	Death with CTE Claims	41
	(a) Death occurred after Final Approval	8
	(b) Complete Claim Package Not Provided	8
	(c) Death with CTE Denial - Death Before 7/7/14 and QD After Final Approval	1
	(d) Special Master Denial	24
2.	ALS Claims	3
	(a) Special Master Denial	1
	(b) Complete Claim Package Not Provided	2
3.	Alzheimer's Disease Claims	72
	(a) Appeals Advisory Panel Denial - Qualifying Diagnosis	42
	(b) Appeals Advisory Panel Denial - Inappropriate Physician	15
	(c) Special Master Denial	10
	(d) Appeals Advisory Panel Denial - Inappropriate Physician	6
	(e) Duplicate Claim Alleging Same Qualifying Diagnosis	2
	(f) Disqualified Physician	1
4.	Parkinson's Disease Claims	11
	(a) Appeals Advisory Panel Denial - Qualifying Diagnosis	6
	(b) Appeals Advisory Panel Denial - Inappropriate Physician	4
	(c) Special Master Denial	1
	(d) Complete Claim Package Not Provided	2
5.	Level 2 Claims	185
	(a) Appeals Advisory Panel Denial - Qualifying Diagnosis	86
	(b) Special Master Denial	30
	(c) Complete Claim Package Not Provided	36
	(d) Physician Disqualified by Special Master	17
	(e) Appeals Advisory Panel Denial - Inappropriate Physician	7
	(f) Disqualified Neuropsychologist	1
	(g) Appeals Advisory Panel Denial - MAF Oversight	12
	(h) Appeals Advisory Panel Denial - Special Assignment	2
	(i) BAP Diagnosis of No Neurocognitive Impairment	1
6.	Level 1.5 Claims	361
	(a) Appeals Advisory Panel Denial - Qualifying Diagnosis	200
	(b) Complete Claim Package Not Provided	77
	(c) Special Master Denial	46
	(d) Appeals Advisory Panel Denial - Inappropriate Physician	18
	(e) Disqualified Physician	11
	(f) Appeals Advisory Panel Denial - MAF Oversight	18
	(g) Disqualified Neuropsychologist	2
	(h) Appeals Advisory Panel Denial - Special Assignment	2
	(i) Untimely Claim Package - Pre-2/6/17 Qualifying Diagnosis	0
7.	No Qualifying Diagnosis - Complete Claim Package Not Provided	211
	(a) Complete Claim Package Not Provided	203
	(b) Special Master Denial	5
	(c) Untimely Claim Package	3
8.	TOTAL DENIAL REASONS PROVIDED ON NOTICES	912
9.	TOTAL NUMBER OF SETTLEMENT CLASS MEMBERS RECEIVING A DENIAL NOTICE	884

NFL CONCUSSION SETTLEMENT

IN RE: NATIONAL FOOTBALL LEAGUE PLAYERS' CONCUSSION INJURY LITIGATION
No. 2:12-md-02323 (E.D. Pa.)

SECTION 11

MONETARY AWARD PAYMENTS

	Status by Confirmed Qualifying Diagnosis	Number	Amount
1.	Notices of Monetary Award (Dollars Shown are Before Holdbacks)	984	\$690,263,579
	(a) Death with CTE	79	\$93,238,543
	(b) ALS	39	\$98,327,542
	(c) Alzheimer's Disease	303	\$137,219,759
	(d) Parkinson's Disease	134	\$85,047,514
	(e) Level 2.0 Neurocognitive Impairment	148	\$124,291,959
	(f) Level 1.5 Neurocognitive Impairment	281	\$152,138,262
2.	Not Ready to be Included on the Next Monthly Payment List; or a Claim Hold is in Place (Dollars Shown are Before Holdbacks)	50	\$44,938,569
	(a) Death with CTE	0	\$0
	(b) ALS	0	\$0
	(c) Alzheimer's Disease	8	\$2,039,998
	(d) Parkinson's Disease	3	\$3,102,253
	(e) Level 2.0 Neurocognitive Impairment	13	\$20,136,823
	(f) Level 1.5 Neurocognitive Impairment	26	\$19,659,496
3.	Ready to be Included on the Next Monthly Payment List (Dollars Shown are Before Holdbacks)	1	\$136,668
	(a) Death with CTE	0	\$0
	(b) ALS	0	\$0
	(c) Alzheimer's Disease	0	\$0
	(d) Parkinson's Disease	0	\$0
	(e) Level 2.0 Neurocognitive Impairment	1	\$136,668
	(f) Level 1.5 Neurocognitive Impairment	0	\$0
4.	Claims in the Payment Process (Dollars Shown are After Holdbacks)	87	\$47,008,110
	(a) Death with CTE	2	\$416,037
	(b) ALS	2	\$140,413
	(c) Alzheimer's Disease	21	\$6,211,492
	(d) Parkinson's Disease	9	\$4,836,592
	(e) Level 2.0 Neurocognitive Impairment	17	\$11,706,786
	(f) Level 1.5 Neurocognitive Impairment	36	\$23,696,791
5.	Paid Claims/Paid to or on Behalf of Class Members or to Lien Holders ¹	1,028	\$565,302,388
	(a) Death with CTE	77	\$88,750,541
	(b) ALS	37	\$91,903,967
	(c) Alzheimer's Disease	274	\$121,688,697
	(d) Parkinson's Disease	122	\$73,635,447
	(e) Level 2.0 Neurocognitive Impairment	117	\$85,822,854
	(f) Level 1.5 Neurocognitive Impairment	219	\$102,707,793
	(g) Derivative Claimants	182	\$793,090

¹ The dollars shown reflect payments issued to Settlement Class Members and on their behalf after all applicable deductions (e.g., Common Benefits Fees, lien holdbacks, potential rescission amounts for Third-Party Funders, etc.).